

Cholera in Canada

To the editor: I read with interest the recent articles on cholera in the Journal (*Can Med Assoc J* 115: 377, 393, 397, 401; 1976) but was surprised to find that the case of cholera in Kingston, Ont. was described as the "first case of cholera to be diagnosed in Canada". Unfortunately, I have no access to facilities for a proper search of relevant literature but MacDermot¹ referred to epidemics of cholera in Montreal in 1831, with 4000 deaths, 1834 and 1849. Jenkins² described attempts, again in Montreal, to prevent cholera from spreading in the summer of 1833, when 1904 of 4420 patients who had cholera died. He also mentioned a smaller epidemic in 1854.

Cholera, therefore, was a serious problem in Canada in the 19th century.

It is a compliment to present-day sanitation and public health that a single case of cholera in Kingston, Ont. merits three articles!

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To the editor: Dr. Lester is correct in stating that the case of cholera reported in the Journal was not the first case to be diagnosed in Canada. In my editorial I should have said the first recent case. Cholera, together with other major endemic diseases such as malaria, typhus and smallpox, was a major cause of illness and death in Canada in the 19th century.

The first recorded cholera pandemic was in 1817 and was confined to Asia and parts of Africa. The second pandemic started in 1826 in India and by 1832 had spread all over Europe. Cholera reached Canada in 1831 as a ship-borne disease and continued to cause outbreaks in Quebec and Montreal for several years. The worst outbreak was in 1833 in Montreal.

The third pandemic started in 1846, and for the next 60 years or so cholera continued to be a problem in North America — partly because of a high rate of immigration of poor, often starving, Europeans, travelling in overcrowded ships with the lowest standards of sanitation.

Classic rice-water stools and tremendous fluid loss, coupled with the high mortality, made differential diagnosis relatively easy prior to the establishment of microbiologic confirmation. Koch demonstrated the cholera vibrio in 1883-84 and by 1900 microbiologic confirmation of outbreaks was common.

I thank Dr. D.H. Starkey for providing me with some of the historical data and Dr. Lester for drawing my attention to

the history of my own hospital.¹

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Intravesical knotting of a urethral "catheter"

To the editor: Foreign bodies removed from various body orifices have often been bizarre, but intravesical knotting of a plastic tube introduced through the urethra by a patient has been reported only once — by Amar in this journal in 1969.¹ I recently encountered a similar case.

A 59-year-old man was seen in the emergency department with a short length of black rubber tube protruding from his external urethral meatus. He claimed he had inserted the tube, the insulating layer of an electric cable, 24 hours earlier to relieve an episode of acute urinary retention and had been unable to remove it subsequently when he obtained relief.

A similar episode of retention 6 months prior to this admission had been treated by a general practitioner by catheterization. The patient's subsequent minimal symptoms of prostatism had not been deemed worthy of further investigation.

Physical examination revealed no abnormality apart from the presence of the tube; the patient's prostate was of normal size. Attempts to remove the tube by traction were painful and unsuccessful, so a radiograph of the bladder was taken (Fig. 1); this revealed a knot in the tube, which was impacted in the prostatic urethra.

A general anesthetic was administered and the tube removed by firm traction. It proved to be 22 cm long with an external diameter of 4 mm. A Foley catheter was inserted; when this was removed 48 hours later the patient was able to micturate satisfactorily. A subsequent intravenous urogram revealed minimal resid-

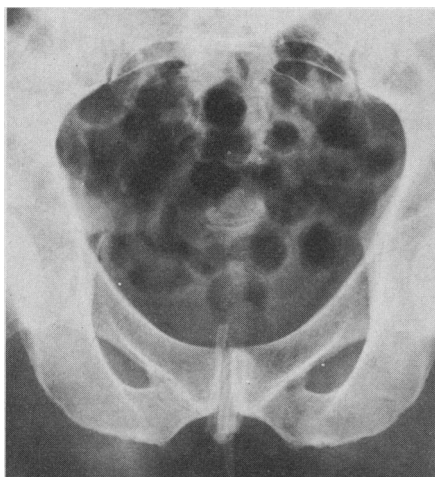


FIG. 1—Knotted tube impacted in prostatic urethra.

ual urine following micturition. The patient is at present under outpatient observation.

Case reports of urethral and intravesical foreign bodies abound in the literature.² Although Campbell³ asserted that sexual aberration is common in the patients and that autoeroticism is fundamental, this patient claimed merely reluctance to disturb his doctor.

I thank Mr. G.S. Makin for permission to report this case.

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Hyperglycemia-induced hyponatremia

To the editor: I have a few comments about the interesting paper on hyponatremia produced by hyperglycemia by Roscoe and colleagues (*Can Med Assoc J* 112: 452, 1975).

The authors' calculations are pertinent to the stage of development of hyperglycemia. However, in clinical practice one is usually faced with the stage of correction of hyperglycemia. During this stage exogenous insulin is available and glucose is taken up by "insulin-sensitive" as well as "insulin-insensitive" tissues. If a fraction of the glucose entering insulin-sensitive tissues remains osmotically active the magnitude of the osmotic water shift should be affected.

For example, in the authors' hypothetical patient (plasma glucose concentration, 986 mg/dl; serum sodium concentration, 128 mmol/l; extracellular fluid [ECF] volume, 15.27 l; intracellular fluid volume in insulin-insensitive tissues [ICFⁱ] plus ECF, 20.08 l; and ICF volume in insulin-sensitive tissues [ICF^s], 19.97 l) if 100 out of 1000 mOsm of glucose disappearing from the ECF space remain osmotically active in the ICF^s space, the total osmolar content of the body will be 12 070 mOsm (12 970 - 900); body fluid osmolarity, 287.4 mOsm/l (12 070 ÷ 42); ICF^s osmolar content, 6267 mOsm (6167 + 100); ICF^s volume, 21.81 l (6267 ÷ 287.4); fluid shift, 1.84 l (21.81 - 19.97); ICFⁱ plus ECF, 18.24 l (20.08 - 1.84); part of fluid shift coming from ECF space, 1.40 l (1.84 x 15.27 ÷ 20.08); final ECF volume, 13.87 l (15.27 - 1.40); final sodium concentration, 141.3 mmol/l (1960 ÷ 13.87); and final plasma glu-

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Precautions: Drugs with atropine-like action should not be used simultaneously with Maxeran since they have a tendency to antagonize effect of this drug on gastrointestinal motility. Maxeran should not be used in conjunction with potent ganglioplegic or neuroleptic drugs since potentiation of effects might occur.

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The recommended dosage of Maxeran should not be exceeded since a further increase in dosage will not produce a corresponding increase in the clinical response. The dosage recommended for children should not be exceeded.

Contraindications: Maxeran should not be administered to patients in combination with MAO inhibitors, tricyclic antidepressants, sympathomimetics and foods with high tyramine content, since safety of such an association has not yet been established. As a safety measure, a two-week period should elapse between using Maxeran and administration of any of these drugs.

The safety of use of Maxeran in pregnancy has not been established. Therefore Maxeran should not be used in pregnant women, unless in the opinion of the physician the expected benefits to the patient outweigh the potential risks to the fetus.

Dosage and administration:

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Adults

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Liquid: 5 — 10 ml (5 — 10 mg) three or four times a day before meals.

Injectable: When parenteral administration is required, 1 ampoule (10 mg) I.M. or I.V. (slowly) to be repeated 2 or 3 times a day if necessary.

Children:

(5 to 14 years): 2.5 to 5 ml (2.5 — 5 mg) three times a day before meals.

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cose concentration, 5.55 mOsm/l (101.3 ÷ 18.24) or 100 mg/dl. Thus the serum sodium concentration would increase 1.5 mmol/l for every 100 mg/dl decrease in blood glucose concentration; the metabolic correction factor would thus be -1.5 mmol/l sodium per 100 mg/dl glucose.

If in the same example 500 rather than 100 out of 1000 mOsm of glucose lost from the ECF space remained osmotically active in the ICF* space the metabolic correction factor would be -2.37 mmol/l sodium per 100 mg/dl glucose.

One should also consider that correction of hyperglycemia creates forces favouring passive sodium entry into the cells. The effect of this on the metabolic correction factor should be opposite to the effect of glucose entry — that is, the larger the rate of intracellular sodium entry, the smaller the absolute value of the metabolic correction factor.

In our study of four anuric hyperglycemic patients (paper submitted for publication) the metabolic correction factor in each patient was close to that predicted by Katz¹ — 1.6 mmol/l sodium per 100 mg/dl glucose. I believe that during correction of hyperglycemia net solute shifts between ECF and ICF space either are insignificant or offset each other in terms of their effect on the ECF solute ratio. I also believe that the use of a correction factor of 1.6 to predict final serum sodium concentration will be proven correct in most hyperglycemic patients.

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Reference

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Psychotropic drug use among women

To the editor: In her article "Psychotropic drug use among women" (*Can Med Assoc J* 115: 760, 1976) R. Cooperstock confirms the well known statistic that there are two women for every man who comes to us for psychiatric support. Considering the frustrations modern women are faced with it is surprising that the ratio is not much higher. In the past women had a well defined role in society — motherhood. From the time they married until menopause intervened they could, and did, have unlimited babies, an activity sentimentally rewarded by the establishment of Mother's Day.

Three things brought a halt to this

reckless fecundity: (1) the introduction and availability of effective methods of birth control, (2) the growing menace of population explosion and (3) the increased difficulty in raising and educating more than a "nuclear family" due to inflation. As a result, the modern young woman, with her two children safe in kindergarten, finds herself all dressed up with nowhere to go. Entombed in an empty house and restricted to such unpaid menial tasks as making beds, cooking meals and pushing a vacuum cleaner, the woman's life seems aimless and lacking in the satisfaction her husband and brother are achieving in the outside world. This is especially true if, as is often the case, she is a college graduate. She usually begins to have symptoms and therefore comes to us for the succour of capsule and shock.

As a gynecologist I encountered quite a number of young women with pains and aches between the umbilicus and pubic hairs who gave the impression that they were leading lives of "quiet desperation". Many had had operations by witless surgeons. All were taking drugs. The only therapy I found of curative value was to get them interested in some activity that provided the satisfaction lacking in their lives. I often had to urge them to break out of their domestic prisons and find paying jobs rather than spend their time doing the family wash and keeping the toilet-bowl clean. Ms. Cooperstock seems to agree with this therapy: "Contemporary women filling numerous roles have somewhat less illness and take fewer tranquilizers and sleeping medications than women filling the 'traditional' female role of housewife."

Occasionally this seemed to work the sort of miracle Jimmy Carter would describe as being "born again", but women found the outside world somewhat unfriendly. They were received largely because the market needed them but they were still in effect second-class citizens. The jobs they filled were usually well down the prestige ladder and the pay was less than men's for doing the same work. While women did get a whiff of freedom and a sense of contributing, their lives were still orchestrated by the "male mystique".

I think it can safely be said that until women find themselves more equitably placed in the working world and they cease to be regarded, by themselves and others, as second-class citizens, the ratio of women to men seeking emotional props from our profession will remain unchanged from its present 2:1.

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